Stress and Poor Physical and Mental Health among Postpartum Mexican American Women: A Test of Heart Rate Variability in Promoting Resilience

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ABSTRACT

Low-income Mexican American women face significant risk for poor health during the postpartum period. Chronic stressors are theorized to negatively impact mental and physical health outcomes. However, physiological factors associated with increased self-regulatory capacity, such as resting heart rate variability, may buffer the impact of stress. In a sample of 322 low-income Mexican American women (mother age 18-42; 84% Spanish-speaking; modal family income $10,000-$15,000), the interactive influence of resting heart rate variability and three chronic prenatal stressors (daily hassles, negative life events, economic stress) on maternal cortisol output, depressive symptoms, and self-rated health at 12 weeks postpartum was assessed. The hypothesized interactive effects between resting heart rate variability and the chronic prenatal stressors on the health outcomes were not supported by the data. However, results showed that a higher number of prenatal daily hassles was associated with increased postpartum depressive symptoms, and a higher number of prenatal negative life events was associated with lower postpartum cortisol output. These results suggest that elevated chronic stress during the prenatal period may increase risk for poor health during the postpartum period.

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Stress and Poor Physical and Mental Health among Postpartum Mexican American Women: A Test of Heart Rate Variability in Promoting Resilience

Although the time period surrounding pregnancy and childbirth often brings happiness and positivity, this life transition can also pose risks to a woman's physical and mental health. Women may experience adverse health in the postpartum period, including increased reports of feeling ill, headaches, and infections (Gennaro & Rosen Bloch, 2005), postpartum depression (Segre, O'Hara, Arndt, & Stuart, 2007), and childbirth-related posttraumatic stress disorder (Bailham & Joseph, 2003).

Poor postpartum health outcomes may partially result from chronic stress during pregnancy and in the postpartum period. Pregnancy-related anxiety (e.g. regarding the course of the pregnancy, the health of the baby, and labor and delivery; Rini, Dunkel-Schetter, Wadhwa, & Sandman, 1999), as well as stress due to weight gain and/or negative body image, monetary stressors, and family stressors (Arizmendi & Affonso, 1987), may develop or intensify during this time period. After childbirth, mothers may face new chronic stressors: disrupted sleep, reduced personal time, and anxiety centered on raising a child (Arizmendi & Affonso, 1987). Health risks may be intensified for low-income Mexican American women, as they may also experience stress related to discrimination, acculturation, immigration status, and poverty, both during pregnancy (DeMarco, Thorburn, & Zhao, 2008) and throughout their lives (Gallo, Penedo, Espinosa de los Monteros, & Arguelles, 2009). Mexican American women also face higher risk than majority group women for poor mental and physical health outcomes such as postpartum depression (Liu & Tronick, 2013), gestational and non-gestational diabetes

(Centers for Disease Control, 2013; Thorpe et al., 2005), and obesity (CDC, 2013). Preventing poor health outcomes during the postpartum period will require a fuller understanding of the ways in which chronic stress impacts physical and mental health, as well as identification of factors associated with resilience in the face of chronic stress.

Stress and Health: The Allostatic Load Model

In general, chronic stress predicts poorer physical and mental health in a wide variety of areas, including cardiovascular disease, the common cold, depression, and negative mood (Cohen et al., 1998; Delongis, Folkman, & Lazarus, 1988; Revollo, Qureshi, Collazos, Valero, & Casas, 2011; Siegrist, 2008; Kivimaki et al., 2013). A commonly accepted model of the relation between chronic stress and health is the allostatic load model (McEwen & Steller, 1993). This model proposes mechanisms through which chronic stress can 'get under the skin' and lead to poor health outcomes. Allostasis refers to the process through which the body attempts to maintain physiological stability in the face of environmental demands (stressors) via coordinated allostatic responses throughout the body (e.g. release of hormones to promote metabolism). Allostatic responses are adaptive in the short run, as they help to maintain physiological stability; however, as stress becomes chronic and allostatic responses are activated repeatedly, these responses may become dysregulated and cause 'wear and tear' on the body. This 'wear and tear', commonly referred to as allostatic load, ultimately degrades mental and physical health (Juster, McEwen, & Lupien, 2010).

The release of cortisol by the hypothalamic-pituitary-adrenal (HPA) axis is one example of an allostatic response. Cortisol is a glucocorticoid hormone that prepares the

body to attend to stressors (e.g. fight or flee) through a variety of actions, including increasing the amount of glucose sent to the muscles, decreasing activity in the immune system, and aiding in the speedy metabolism of fats, proteins, and carbohydrates to summon energy reserves for action.

Consistent with the allostatic load model, chronic stress can lead to dysregulated levels or responses of cortisol (Miller, Chen, & Zhou, 2007). In some cases, chronic stress leads to a *blunting* of cortisol levels in the body, as the body can no longer muster an adequate cortisol response to stressors. Conversely, chronic stress might instead lead to an *increase* of cortisol in the body, either by releasing a larger amount of cortisol than necessary to respond to environmental demands, or by failing to appropriately halt the stress response after the stressor ends, thus increasing overall cortisol output. Although the antecedents of these divergent patterns of cortisol dysregulation are not yet fully understood, particular types of stress may lead to unique patterns of cortisol dysregulation. Meta-analyses investigating the relation between stressor type and cortisol dysregulation have suggested that ongoing chronic stressors generally lead to an increase in cortisol output, whereas more temporally distal stressors (e.g. stress during early development) lead to blunted cortisol output (Miller, Chen, & Zhou, 2007). Both blunted and exaggerated cortisol levels play a role in models of risk for physical and mental disease, including metabolic syndrome (Bjorntorp & Rosmond, 1999), cardiovascular disease (Manenschijn et al., 2013), and depression (McEwen, 2000).

Heart Rate Variability

Many individuals are able to remain resilient despite experiencing chronic stress (Luecken & Gress, 2010; Luthar, Sawyer, & Brown, 2006). In the context of stress and allostatic load, resilience refers to the ability to respond to environmental demands efficiently through adaptive stress response engagement and termination (Karatsoreos & McEwen, 2011). Heart rate variability may promote physiological resilience in the face of chronic stress. Heart rate variability (HRV) refers to fluctuations in heart rate resulting from autonomic influence on the heart. The autonomic nervous system (ANS) works with other bodily systems to maintain allostasis by responding to environmental demands. The ANS includes two reciprocal systems: the sympathetic (SNS) and parasympathetic (PNS) nervous systems. Whereas the SNS slowly and strongly ramps up bodily processes in response to environmental demands, the PNS works via the vagus nerve to regulate the body through quicker, more refined actions. The vagus nerve, which travels from the nucleus ambiguus region in the brain to enervate the heart, works like a 'brake', preventing the heart from beating at the very fast rate that the heart's pacemaker, the sinoatrial node, sets. However, as environmental demands change such that a faster heart rate would be more adaptive, the vagus nerve partially releases the 'brake', allowing the heart to beat faster. As the vagus nerve creates a slower or faster heart rate in response to constantly shifting environmental demands, heart rate becomes more variable- thus the term 'heart rate variability' (Applehans & Luecken, 2006).

Heart rate variability can be measured via either resting or reactive HRV. Measures of resting HRV occur over a time period in which conditions remain consistent

and the individual rests (e.g. lying calmly on a sofa; Sandercock, Bromley, & Brodie, 2005). Resting HRV has demonstrated high levels of consistency among healthy individuals over time intervals of a year or more (Burleson et al., 2003; Sandercock et al., 2005). Due to this consistency, resting HRV is conceptualized as reflecting a trait-like index of an individual's capacity to regulate their heart rate in response to varying environmental demands (Bertsch, Hagemann, Naumann, Schachinger, & Schulz, 2012; Thayer, Hansen, Saus-Rose, & Helge Johnsen, 2009). Reactive HRV, on the other hand, refers to measurements of heart rate over a time period in which an individual reacts to a challenging stimulus (e.g. the Trier Social Stress Task; Sandercock et al., 2005). The current literature review and study will focus on resting HRV.

Lower resting HRV predicts poorer health outcomes, including increased risk for all-cause mortality and morbidity (Thayer & Lane, 2007). More specifically, low resting HRV predicts adverse cardiovascular events (Hillebrand et al., 2013), and resting HRV correlates inversely with risk for hypertension (Liao et al., 1996). Depressed individuals have lower resting HRV than healthy controls (Rottenberg, Clift, Bolden, & Salomon, 2007), and individuals with lower resting HRV report more anxiety symptoms than those with higher resting HRV (Piccirillo et al., 1997). High resting HRV may be associated with better health outcomes through several different mechanisms; one possibility is that high resting HRV promotes an individual's physiological capacity to efficiently regulate their emotions, thus minimizing the emotional or physical toll of stress.

Emotion regulation involves monitoring, evaluating, and modifying of emotional responses to match the context in which they occur, so as to maximize adaptive responses

(Applehans & Luecken, 2006); for example, successful emotion regulation might involve controlling one's feelings of frustration. Poor emotion regulation predicts risk for numerous mental health disorders, including depression, anxiety, and substance use (Berking & Wupperman, 2012), as well as physical health disorders, including coronary heart disease and metabolic syndrome (Kinnunen, Kokkonen, Kaprio, & Pulkkinen, 2005; Kubzansky, Park, Peterson, Vokonas, & Sparrow, 2011). The vagus nerve may have evolved to support emotion regulation when early humans became a more social species, and reproductive success began to hinge on effectively regulating emotional and social responses (Porges, 1997). Although the evolutionarily older SNS substrates also react to environmental demands, they do so at a slower rate, making SNS reactions better suited to straightforward fight or flight responses than to regulation of emotional and social responses. The vagus nerve not only facilitates nuanced emotional and social responses via increased heart rate variability, but it also enervates muscles in the face, larynx, and other cranial areas involved in emotional expression, listening, and other social behaviors. This vagal connection between the brain, heart, and face allows for coordinated regulation of heart rate and emotional responses to meet environmental demands (Porges, 1997).

Researchers have empirically investigated the relation between resting HRV and capacity for emotion regulation. Higher resting HRV predicts better recognition of emotion in others, one aspect of emotion regulation (Quintana, Guastella, Outhred, Hickie, & Kemp, 2012). Additionally, the relation between resting HRV and well-being is partially mediated through the habitual use of executive emotion regulation strategies (e.g. planning, reappraisal; Geisler, Vennewald, Kubiak, & Weber, 2010). Executive

functioning, which measures an individual's ability to self-regulate emotion, cognition, and attention via the prefrontal cortex (Miyake & Friedman, 2012), is also related to resting HRV; individuals with higher resting HRV perform more quickly and accurately on tasks requiring high levels of executive function than individuals with lower resting HRV. Resting HRV does not, however, predict performance on tasks which do not require high levels of executive function. This may suggest that, rather than enhancing performance indiscriminately, resting HRV specifically enhances performance in situations requiring high levels of executive function and self-regulation (Hansen, Johnsen, & Thayer, 2003).

Resting HRV also predicts the use of more effective stress coping strategies. Resting HRV levels correlate significantly with use of engagement coping; the higher an individual's resting HRV levels, the more they report using engagement strategies (e.g. social support seeking, situation control) to cope with stress (Geisler, Kubiak, Siewert, & Weber, 2013). Fabes & Eisenberg (1997) found that higher resting HRV was associated the use of more constructive coping strategies in the face of everyday stressors. However, this association only held for stressors which individuals rated as moderately or highly intense, indicating that when there are few environmental demands that an individual must to respond to, or when these demands are not intense, HRV is not associated with better coping.

Previous research has shown that not only does higher resting HRV predict better emotion regulation and more use of effective coping strategies, but that resting HRV also supports biological resilience in the face of stress. For example, among individuals faced

with a social stress task, high resting HRV was significantly correlated with both a larger cardiac stress response (i.e. increased heart rate and reduced HRV) and a quicker recovery after stress (i.e. speedier and larger increase in HRV and decrease in heart rate back to pre-stress levels; Souza et al., 2007; Souza et al., 2013). This indicates that individuals with high resting HRV are better able to both mount and end an adaptive cardiac stress response in reaction to environmental demands.

High resting HRV may also moderate the impact of chronic stress on cortisol output. After undergoing a laboratory stressor (i.e. a mental arithmetic task) which elicits a cortisol stress response, individuals with high resting HRV return more quickly to pre-stressor cortisol levels than those with low resting HRV, which indicates a more adaptive termination of the cortisol stress response (Weber et al., 2010). As noted previously, dysregulation in the termination of the cortisol stress response has been implicated as a factor in the development of allostatic load (e.g. dysregulated cortisol levels; Miller et al., 2007) and poor mental and physical health outcomes (Bjorntorp & Rosmond, 1999 ; Manenschijn et al., 2013; McEwen, 2000) after chronic stress. In a study of the impact of chronic stress on stress reactivity, among children exposed to high chronic stress (i.e. a disorganized household), only those with low resting HRV had elevated levels of cortisol; those with high resting HRV had no difference in cortisol output as a function of increased chronic stress (Blair, Berry, Mills-Koonce, & Granger, 2013).

In combination, prior research suggests that high resting HRV indicates underlying biological resilience. As adaptive biological stress responses are associated with better mental and physical health (Berking & Wupperman, 2012; Bjorntorp &

Rosmond, 1999; Kinnunen et al., 2005; Kubzansky et al., 2011; Manenschijn et al., 2013; McEwen, 2000), resting HRV may also moderate the impact of chronic stress such that among those experiencing chronic stress, those with high resting HRV may experience less depressive symptomatology and better self-rated health than those with low resting HRV. If high resting HRV does indeed promote adaptive cortisol responses and levels, less depressive symptomatology, and better self-rated health in the face of chronic stress, this has particularly important implications for populations at increased risk for poor physical and mental health outcomes, such as pregnant and postpartum Mexican American women.

The Current Study

The current study will investigate stress, heart rate variability, and markers of mental and physical health in a sample of low-income Mexican American women. The women were recruited during pregnancy as part of a larger study, *Las Madres Nuevas*, and followed through the 3rd postpartum year. I hypothesize that resting HRV will moderate the relation between prenatal chronic stress and three postpartum outcomes: cortisol output, postpartum depression, and self-rated health. Across the sample, higher prenatal stress is expected to predict elevated cortisol output, elevated depressive symptomatology, and poorer self-rated health at 3 months postpartum. However, the association of chronic stress with poor postpartum outcomes is expected to be attenuated among women with higher resting HRV.

Method

Participants

Participants included 322 women (mean age = 27.84, SD = 6.5) and their infants (see table 1 for demographics). For the larger study, data was collected prenatally (26-38 weeks gestation), and at 3, 6, 12, 15, 18, 21, and 24 weeks postpartum. The current analyses primarily use data from the prenatal, 6, and 12 week time points. Additionally, data from the 18 week time point are used for missing data analyses. Women were recruited from a hospital-based prenatal clinic that serves low-income women from the surrounding community. Eligibility criteria included: 1) self-identification as Mexican or Mexican American, (2) fluency in English or Spanish, (3) age 18 or older, (4) low-income status (family income below $25,000 or eligibility for Medicaid or Federal Emergency Services coverage for the birth), and (5) anticipated delivery of a singlet baby with no prenatal evidence of a serious health or developmental problem.

Recruitment and retention

During prenatal care appointments, women were approached by a female, bilingual interviewer who explained the study and assessed eligibility. Of women who were eligible, 56% agreed to a home visit between 26-38 weeks gestation (mean 35.4 weeks, SD = 2.8) at which informed consent was obtained. To minimize participant burden, the study followed a "planned missingness" design: all participants were assigned to complete the prenatal and six week visits, but a random 2/3 of the sample were assigned to complete the 12 and 18 week visits. Random assignment was determined by a computer algorithm conducted prior to the first data collection. Of the 323 women who

consented to the study at the prenatal visit, 312 (96%) completed a 6-week visit. Of the random 2/3 of the sample assigned to complete the 12 week visit, 205 mothers participated (95%), and of the random 2/3 of the sample assigned to complete the 18 week visit, 209 mothers participated (93%).

Procedure

All interviews were conducted at participants' homes in their choice of Spanish (85%) or English (15%). Due to variability in literacy, survey questions were read aloud to all participants. Women were given visual aids with written and graphic descriptions of item response formats. At the 6, 12, and 18 week interviews, salivary cortisol samples were collected from mothers before and after the mother-infants dyads participated in video-recorded interaction tasks that began approximately 30 minutes after arrival in the home. At the 6 week interview, heart rate data was recorded during a 7-minute resting baseline period using Trillium ECG monitors (Forest Medical, LLC; East Syracuse, NY, USA). The total duration of the home visits was approximately 2 hours. Women were compensated $75 and small gifts (e.g., bath oils) at the prenatal interview, and $50 and small gifts (e.g., bibs, rattles) at the 6, 12, and 18 week interviews.

Measures

Questionnaires. Prenatal chronic stress was measured using three different self-report measures of stress, obtained during prenatal interviews. These three stress scales (Negative Life Events, Daily Life Hassles, Economic Hardship) were chosen to represent a variety of chronic stressors salient for a low-income sample.

Negative life events. Negative life events were measured using a 13-item scale from the Pregnancy Risk Assessment Monitoring System (PRAMS; Centers for Disease Control, 2009) from which a count of negative life events was obtained. Participants were asked to respond with either "true" or "false" with regards to if a negative life event (e.g. "You separated or divorced from your husband or partner") had occurred to them over the last 12 months. Higher scores indicate that a greater number of negative life events occurred over the last 12 months.

Daily Life Hassles. Daily life hassles were measured using a 25-item scale (Kanner, Coyne, Schaefer, & Lazarus, 1981). Participants were asked if a daily hassle (e.g. making thoughtless, inconvenient mistakes) had happened to them in the past month. Higher scores reflect a greater frequency of daily hassles. Among our sample, this scale was found to be reliable (α=0.90).

Economic Hardship. Economic hardship was measured using a 20-item scale (Barrera, Caples, & Tien, 2001). Participants were asked first to think about the next three months, and were asked, for example, "How often do you expect that you and the family members in your household will have to do without the basic things that your family needs?" Participants then responded on Likert scale ranging from 1 (almost never or never) to 5 (almost always or always). After answering all questions with regards to the next three months, participants then responded to the same items, but instead were asked to think back to the previous three months. The scale includes subscales: not enough money for necessities, economic adjustments/cutbacks, and financial strain. After raw scores for these subscales were obtained, these subscale scores were transformed into

z scores and then summed to create a composite score of economic hardship. Higher scores reflect higher economic hardship. Among our sample, this scale was found to be reliable (α=0.72).

Depressive Symptoms. 10 item Edinburgh Postnatal Depression Scale (Cox, Holden, & Sagovsky, 1987) was given prenatally and at six, twelve, and eighteen weeks postpartum; depressive symptomatology at the 12 week visit will be used as a dependent variable, with data from the prenatal time point serving as a covariate, and data from the 6 week and 18 week visits being used as part of the missing data analyses. The EPDS has been validated in English (Cox et al. 1987) and Spanish (Garcia-Esteve, Ascaso, Ojuel, & Navarro, 2003). Higher scores reflect higher depressive symptomatology. Among our sample, this scale was found to be reliable (α=0.82 at prenatal, 0.86 at six weeks, 0.84 at twelve weeks, and 0.85 at eighteen weeks).

Self-Rated Health. Self-rated health was assessed prenatally and at six, twelve, and eighteen weeks postpartum; self-rated health at the 12 week visit will be used as a dependent variable, with data from the prenatal time point serving as a covariate, and data from the 6 week and 18 week visits being used as part of the missing data analyses. Self-rated health was measured using a single item, "Overall, your health status is ____." Participants responded using a five-point Likert scale ranging from "Poor" up to "Excellent." Single-item measures of self-rated health have been demonstrated to be stable in adults, and have been found to be predictive of specific health outcomes, general reports of long-standing illness, health-related behavior, and psychological distress (Hertzman, Power, Matthews, & Manor, 2001).

Mother-infant Interaction Task. A series of five structured video-recorded mother-infant observational episodes were conducted. Episodes were selected to elicit mild frustration for the mother and the infant. The infant was in an infant seat with the mother seated directly across from him/her. The episodes included 1) Free play (5 minutes), 2) Arm Restraint (2 minutes), 3) Soothing (3 minutes), 4) Teaching task (5 minutes; mothers are asked to "teach" her child a task from the Bayley Scales of Infant Development II (Bayley, 1993) that reflects a skill 1-2 months beyond the infant's capabilities), and 5) Peek-a-boo (3 minutes).

Heart Rate Variability. Mother heart rate data were recorded at 256 Hz with electrocardiography (ECG) equipment from Forest Medical, LLC (Trillium 5000; East Syracuse, NY, USA) for a 7-minute baseline period, as well as throughout the mother-infant interaction tasks, at the 6 week visit. During the baseline period, mothers and infants were seated upright at rest. Mothers were instructed to "please relax quietly for the next seven minutes and breathe normally…the monitor is sensitive to movement and speaking, so please sit quietly without moving for a few minutes." Electrodes were placed on participants' left shoulder and right and left waist in a standard three-lead configuration.

Resting respiratory sinus arrhythmia (RSA) during the first 5 minutes of this 7-minute baseline was calculated as a measure of resting HRV. Resting RSA indexes oscillations in heart rate produced by respiration, which are mediated by parasympathetic influences on the heart; the sympathetic nervous system's speed of action on the heart is too slow to provoke an influence on the heart that covaries with respiration. QRSTool

software 1.2.2 (Allen, Chambers, & Towers, 2007) was used to process the ECG data and automatically obtain R-spikes from the ECG data. QRSTool software was then used by trained coders to manually correct misidentified or unidentified R-spikes, and obtain R-R interval data. Using CardioBatch software (Brain-Body Center, 2007), a moving polynomial filter was then applied to the R-R interval data to extract heart rate variability in the frequency band of RSA (for adults, 0.12-0.40 Hz). The resting RSA estimates from this analysis were log-transformed, and a mean resting RSA value averaged from 30-second epochs was obtained.

Cortisol sampling. Saliva samples were obtained from mothers before and after the mother-infant interaction tasks at the 6, 12, and 18 week home visits; cortisol at the 12 week visit will be used as a main outcome variable, with data from the 6 week and 18 week visits being used as part of the missing data analyses. Cortisol samples were obtained immediately prior to the first task, and at 0, 20, and 40 minutes post-task, using the Salivette sampling device (Sarstedt, Rommelsdorf, Germany). Saliva samples were frozen and mailed on dry ice to Salimetrics (State College, PA) where they were assayed for free cortisol. Cortisol levels were indexed by cortisol area under the curve with respect to ground (AUCg) using the trapezoid formula (Pruessner, Kirschbaum, Meinlschmidt, & Hellhammer, 2003). Cortisol AUCg was calculated using cortisol values and time of collection for each of the 4 cortisol samples, and then log-transformed (base 10) to correct for deviations from normality. The time period in which the cortisol samples were collected ranged from 55 to 79 minutes across participants. Because longer collection periods will result in a higher cortisol AUCg values due to differences in

collection period length, cortisol AUCg was multiplied by a ratio of individual collection period length over sample average collection period length.

Results

Data cleaning

Raw cortisol values and cortisol AUCg values were inspected for potential outliers, including values above normal physiological levels (45 nmol/l), or values over three standard deviations above the sample mean. Cortisol values over three standard deviations above the sample mean were judged for inclusion based on how distant they were from the rest of the scores, extreme changes from one sample to the next that did not fit typical patterns of reactivity, and medical prescriptions and diagnoses that might make the sample unusable (e.g. use of thyroid medication). Cortisol values from four mothers were removed from analyses due to extreme values which were between 6 and 11 standard deviations above the sample mean.

Preliminary analyses

Sample statistics (means, standard deviations, range, skew, and kurtosis) were obtained for all study variables (see Table 2). Zero-order correlations between study variables were also calculated (see Table 3).

Data Reduction. Correlations among the three prenatal stress measures were examined to assess whether any could be combined to form a composite stress score for data reduction purposes. Daily hassles and negative life events were significantly positively correlated (r =.27, *p*=.001), daily hassles and economic stress were

significantly positively correlated (r =.39, *p*=.001), and negative life events and economic stress were significantly positively correlated (r =.32, *p*=.001). However, none were highly correlated (i.e., r >= .40), and so no composite stress scores were formed. Primary analyses were completed separately for each of the three prenatal stress measures.

Cortisol Covariates. Possible covariates for analyses concerning cortisol AUCg included mother's age, education, country of birth, body mass index (BMI), time of cortisol collection, time at which the mother last ate, breastfeeding status, time at which the mother last breastfed, hormonal birth control or other medication use, average and recent caffeine use, and recent exercise. Time of cortisol collection was significantly negatively correlated with cortisol AUCg (r =-.58, *p*=.001), and was included in primary analyses with cortisol AUCg. Recent caffeine use was also significantly negatively correlated with cortisol AUCg (r =-.21, *p*=.004); however, a partial correlation between recent caffeine use and cortisol AUCg controlling for time of cortisol collection was not significant (r=.001, *p*=.996), indicating that the correlation between recent caffeine use and cortisol AUCg was confounded by time of cortisol collection. Thus, recent caffeine use was not included in primary analyses with cortisol AUCg.

Missing resting HRV and cortisol AUCg data. Resting HRV and cortisol AUCg data were examined to assess amount of missing data, reasons for missing data, and whether mothers with missing data differed from those with complete data.

Heart Rate Variability. Of the 322 participants, 39 were missing data for resting HRV at the 6 week visit (12%); 12 missed the time point due to dropping out of the study or difficulty in scheduling during the visit time window, 3 completed the time point but

did not complete the interaction tasks or provide biological data due to mother or infant illness or completing the interview via phone, and 24 completed the time point but were not found to have at least 2 minutes of usable heart rate data, and so no resting HRV values were computed.

T-tests were employed to compare mothers who were missing resting HRV data at the 6 week visit to mothers who had resting HRV data at the 6 week visit; these groups did not significantly differ in religion, country of birth, marital status, number of children, level of education, work status, income, baby gestational age, or mother's age.

Cortisol AUCg. Of the 322 participants, 130 were missing data for cortisol AUCg at the 12 week visit (40%); 111 participants (34%) were not assigned to complete the 12 week visit due to the planned missingness design used by the larger study in order to reduce participant burden. In addition to the planned missingness, an additional 19 participants (6%) had unplanned missing cortisol data: 9 missed the time point due to dropping out of the study or difficulty in scheduling during the visit time window, 3 completed the time point but did not complete the interaction tasks or provide biological data due to mother or infant illness, and 3 had incomplete cortisol data (e.g. only one cortisol sample out of four). Additionally, 4 mothers had raw cortisol values between 6 and 11 standard deviations above the sample mean, and were removed from analyses.

One-way ANOVA tests were employed to compare mothers who had cortisol data at 12 weeks, mothers with planned missing cortisol data at 12 weeks, and mothers with unplanned missing cortisol data at 12 weeks; these groups did not significantly differ in religion, country of birth, marital status, number of children, work status, income,

education level, or baby gestational age. However, the three groups significantly differed in age ($F(2, 318)=5.509$, $p=.004$); mothers with cortisol data were the oldest (28.60 years old), followed by mothers with planned missing cortisol data (27.04), with mothers with unplanned missing cortisol data being the youngest (24.16). A simple contrast comparing mothers with cortisol data to mothers with planned missing cortisol data was significant, $t(261.632)=2.109$, $p=.036$. A simple contrast comparing mothers with cortisol data to mothers with unplanned missing cortisol data was also significant, $t(28.453)= 3.832$, $p=.001$. Finally, a simple contrast comparing mothers with planned missing cortisol data to mothers with unplanned missing cortisol data was also significant, $t(30.410)= 2.440$, $p=.021$. In order to account for these significant differences, mother age was added as a covariate in the primary analyses.

Missing data procedures. Maximum likelihood estimation was conducted in Mplus version 7.11 (Muthén & Muthén, 1998-2011) in order to account for missing data for cortisol and self-rated health. Data were expected to be missing completely at random due to planned missingness procedures used by the larger study in order to reduce participant burden: all participants were assigned to complete the prenatal and six week visits in person. However, only a random 2/3 of the sample were assigned by a computer algorithm to complete the 12 week visit in person. Thus, only 2/3 of the sample has data for cortisol AUCg and self-rated health at the 12 week visit. Depressive symptomatology was collected from the full sample at 12 weeks; the 1/3 of the sample who did not complete the 12 week visit in person reported on depressive symptomatology via phone.

In order to account for planned missing data for cortisol and self-rated health, auxiliary variables were used in these primary analyses (Enders, 2011). These auxiliary variables included cortisol AUCg and self-rated health at the 6 week and 18 week visits. These auxiliary variables were chosen because they were strongly correlated with the study outcome variables (see Table 3).

Regression Diagnostics. In order to examine the potential influence of outliers on the regression analyses, regression diagnostics were employed. DFFITS, DFBETAS, and studentized deleted residuals were examined for each case for each regression analysis; cases with values above thresholds recommended in the literature (e.g. DFFITS=1, DFBETAS=1, studentized deleted residuals=3; Neter, Wasserman, & Kutner) were given closer examination. Cases were removed from the sample only if evidence suggested that their data might be suspect (e.g. taking medication known to alter cortisol medication) or that they were conceptually dissimilar to other participants and should not be analyzed with the rest of the sample (e.g. reported not being of Mexican American origin).

Primary Results

Cortisol. The primary analyses included nine sets of models. In the first set of regression equations, cortisol AUCg at the 12 week visit was entered as the dependent variable, with each of the three prenatal stress measures, resting HRV, and their interactions entered as independent variables, and time of data collection and mother age included as covariates, for a total of three separate regression equations with cortisol AUCg as the dependent variable (see Table 4). Negative life events significantly predicted cortisol AUCg (*B*=-.014, *SE*=.007, *p*=.04), such that a higher number of

negative life events predicted lower cortisol AUCg. However, neither daily hassles, economic stress, resting HRV, nor any of the interactions significantly predicted cortisol AUCg. As the interactions were not predictive of cortisol AUCg, exploratory analyses were run to more directly assess the main effects of daily hassles, negative life events, economic stress, and resting HRV on cortisol AUCg, still controlling for time of data collection and mother age. Again, negative life events were found to significantly predict cortisol AUCg at 12 weeks (B=-.015, SE=.007, p=.028), such that a higher number of negative life events predicted lower cortisol AUCg.

Regression diagnostics revealed one case that exceeded the recommended absolute value of 1 on the measure of DFFITS for all three regression equations containing cortisol (DFFITS values for this case ranged from -1.03 to -1.07); this case also had a studentized deleted residual above the recommended cutoff value of 3 for all three regression equations containing cortisol (studentized deleted residual values for this case ranged from -4.71 to -4.88). Further examination did not reveal information that theoretically supported removing this case from the dataset. Thus, the final regression results in Table 4 include this case.

Depressive symptomatology. In the second set of regression equations, depressive symptomatology at the 12 week visit was entered as the dependent variable, with each of the three prenatal stress measures, resting HRV, and their interaction entered as independent variables, controlling for prenatal depressive symptomatology, for a total of three regression equations with depressive symptomatology as the dependent variable (see Table 5). Daily hassles significantly predicted depressive symptomatology at 12

weeks (*B*=.05, *SE*=.02, *p*=.013); a higher number of prenatal daily hassles was associated with higher depressive symptomatology at 12 weeks. However, neither negative life events, nor economic stress, nor heart rate variability, nor any of the interactions significantly predicted depressive symptomatology at 12 weeks. As the interactions were not predictive of depressive symptomatology, exploratory analyses were run to more directly assess the main effects of daily hassles, negative life events, economic stress, and resting HRV on depressive symptomatology, still controlling for prenatal depressive symptomatology. Again, daily hassles were found to significantly predict depressive symptomatology at 12 weeks (*B*=.049, *SE*=.020, *p*=.015), such that a higher number of daily hassles was associated with higher depressive symptomatology.

Regression diagnostics revealed three cases with studentized deleted residuals above the recommended cutoff value of 3 for all three regression equations containing depressive symptomatology (studentized deleted residual values ranged from 3.33 to 3.84). Further examination did not reveal information that theoretically supported removing these cases from the dataset. Thus, the final regression results in Table 5 include these cases.

Self-rated health. In the third set of regression equations, self-rated health at the 12 week visit was entered as the dependent variable, with each of the prenatal stress measures, resting HRV, and their interaction entered as independent variables, controlling for prenatal self-rated health, for a total of three regression equations with self-rated health as the dependent variable (see Table 6). No study variables significantly predicted self-rated health at 12 weeks. As the interactions were not predictive of self-

rated health, exploratory analyses were run to more directly assess the main effects of daily hassles, negative life events, economic stress, and resting HRV on self-rated health, still controlling for prenatal self-rated health. Again, no study variables significantly predicted self-rated health at 12 weeks.

Regression diagnostics revealed one case with a studentized deleted residuals above the recommended cutoff value of 3 for all three regression equations containing self-rated health (studentized deleted residual values ranged from -3.03 to -3.32). Further examination revealed that this participant had an abnormally large decrease in self-rated health from the prenatal to the 12 week home visit, at which time they reported that they had recently been diagnosed with a gallbladder tumor. This case was removed, and analyses were re-run on an exploratory basis. No changes in the results occurred after removing this case, and thus it is included in the reported results in Table 6.

Discussion

Although the birth of a child is often a time of joy, many women also experience adverse health in the postpartum period; poor health outcomes may partially result from chronic stress (McEwen & Steller, 1993). Health risks are higher for low-income Mexican American women, who may experience higher levels of stress during pregnancy (DeMarco et al., 2008) and across the lifespan (Gallo et al., 2009), and who experience higher rates of poor mental and physical health outcomes during the postpartum period (CDC, 2013; Liu & Tronick, 2013). As low resting heart rate variability (HRV) has been shown to predict poor health (Thayer & Lane, 2007) and high resting HRV has been linked to better ability to regulate emotions and employ coping strategies in the face of stress (Fabes & Eisenberg, 1997), resting HRV was identified as a possible marker of physiological resilience among Mexican American women. The current study investigated the ways in which both major chronic prenatal stressors (i.e. negative life events such as becoming homeless) and day-to-day chronic prenatal stressors (i.e. daily hassles, economic stress) impact the physical and mental health (i.e. cortisol AUCg, depressive symptomatology, self-rated health) of Mexican American women in the postpartum period, and also assessed whether resting HRV might moderate the impact of stress on physical and mental health.

The hypothesized interactions between the prenatal stressors (daily hassles, negative life events, and economic stress) and resting HRV on cortisol AUCg, depressive symptomatology, and self-rated health were not supported by the data. This was unexpected, given resting HRV's relation to emotion regulation (Fabes & Eisenberg,

1997; Geisler et al., 2013), which is associated with fewer mental and physical health problems (Berking & Wupperman, 2012; Kinnunen et al., 2005; Kubzansky et al., 2011) and has previously been shown to moderate the relation between stress and depression in general adult samples (Vanderhasselt et al., 2014). Additionally, previous research among children has demonstrated that resting HRV moderates the impact of chronic stress (chaotic home environment) on cortisol output (Blair et al., 2013). It may be that resting HRV is most protective when more active coping strategies are not usable or for stressors that are uncontrollable. Young children in a chaotic home environment may benefit from a higher physiological capacity to regulate their emotions, as they may lack the cognitive capacity to use other coping strategies and may have limited ability to control their surroundings to reduce stress. Adult women undergoing negative life events or economic stress, on the other hand, may benefit more from strategies such as problem-focused coping, which has previously been demonstrated to moderate the impact of stress on depression during the postpartum period (Terry, Mayocchi, & Hynes, 1996). Although increased resting HRV may support active or problem-focused coping by boosting capacity to regulate emotions and reducing negative affectivity in response to stress (Fabes & Eisenberg, 1997), high resting HRV on its own may not sufficient to protect mothers undergoing chronic stress if they do not have the other resources needed to successfully cope with stress (e.g. social support, problem solving skills). For example, among general samples higher resting HRV predicts fewer depressive symptoms only among individuals who perceive high social support (Hopp et al., 2013).

It may also be the case that factors other than resting HRV more strongly moderate the impacts of stress on postpartum outcomes among low-income MA mothers.

For example, the impact of prenatal economic stress on postpartum cortisol AUCg has previously been shown to be moderated by family support in this sample (Jewell, Luecken, Gress-Smith, Crnic, & Gonzales, in press). Social support has also been found to moderate the effects of life event stress (e.g. moving, the death of a family member) on birth outcomes and postpartum depression (Collins, Dunkel-Schetter, Lobel, & Scrimshaw, 1993). Previous research has indicated that this buffering effect is strongest among low-SES women (Turner, Grindstaff, & Phillips, 1990). For MA women, for whom cultural values of familism emphasize the value of close and supportive family relationships and for whom family support is theorized to be a primary protective factor (Sagrestano, Feldman, Rini, Woo, & Dunkel-Schetter, 1999), social support from families or romantic partners may have a stronger impact on cortisol AUCg, depressive symptoms, and self-rated health than resting HRV.

Additionally, it is possible that resting HRV may not moderate the effects of chronic stress specifically during the perinatal period. Among non-pregnant women, HRV is impacted by progesterone, one of the main hormones implicated in changes during pregnancy. HRV levels are highest just prior to ovulation, at which time progesterone levels are low (Tenan, Brothers, Tweedell, Hackney, & Griffin, 2014). Among pregnant women, HRV (as indexed by respiratory sinus arrhythmia) decreases throughout the third trimester of pregnancy, and then begins to increase again before childbirth, although not quite to 'baseline' levels (DiPietro, Costigan, & Gurewitsch, 2005). This is thought to be due to increases in metabolic demands in the later stages of pregnancy, requiring increased sympathetic activation and decreased parasympathetic activation. However, no previous studies have investigated changes in HRV

longitudinally across the postpartum period, and so it is unclear how HRV levels change during this time. HRV may continue to be attenuated in the postpartum period. Alternatively, the drop in progesterone levels following childbirth may lead to increased HRV during the postpartum period. Regardless, HRV may not be stable during the postpartum period, which may obscure or attenuate the relations between resting HRV and stress, cortisol, depressive symptoms, and self-rated health.

Furthermore, although no previous studies comparing ethnic differences in HRV have included Mexican Americans, there are ethnic differences between White and African American individuals in resting HRV (Choi et al., 2006), and between White, African American, and Chinese American individuals in HRV during sleep (Hall et al., 2013). While HRV is 'trait-like', there are also age-related declines in resting HRV (Sloan et al., 2008). These declines are steeper among lower income individuals (Fuller-Rowell et al., 2013). It may be that resting HRV operates differently among low-income Mexican American women than in other populations.

Despite the lack of support for the hypothesized interaction, results did demonstrate that prenatal daily hassles were predictive of postpartum depressive symptomatology; a higher number of prenatal daily hassles was associated with an increase in depressive symptoms from the prenatal to the postpartum time point. This is in line with prior research demonstrating that a higher number of daily hassles are associated with more depressive symptoms in the postpartum period (Field, Hernandez-Reif, & Diego, 2006; Honey, Morgan, & Bennett, 2003). Prenatal daily hassles did not predict postpartum cortisol levels or self-rated health. It may be that daily hassles

uniquely contribute to depressive symptomatology during the postpartum period. For women already faced with many daily hassles before childbirth, the added stress of a new baby may tax their resources and lead them to feel overwhelmed, leading to an increase in depressive symptoms into the postpartum period. Daily hassles may not, however, be strong enough in magnitude to exert similar effects on cortisol or self-rated health, which may be more impacted by stronger or more proximal stressors.

Results also demonstrated that prenatal negative life events were predictive of postpartum cortisol AUCg. However, results were in the opposite direction as hypothesized: a higher number of prenatal negative life events was associated with lower postpartum cortisol output. Although contrary to the predicted direction of results, theorists have suggested that major traumatic events and stressors are associated with an initial increase in cortisol output, followed by a downregulation of the cortisol response and subsequently lower cortisol levels (Miller et al., 2007). Although major negative life events may have lasting effects and be considered chronic stressors, the magnitude of the stressor may differentially influence later patterns of cortisol output. Additionally, given the low socioeconomic status of the study participants, they are likely to have experienced significant hardship (Brady & Matthews, 2002); this long-standing stress may have led to blunted cortisol responses and lower cortisol levels by this point in these mothers' lives. As lower levels of cortisol have been associated with postpartum depression (Burke, Fernald, Gertler, & Adler, 2005; Jolley, Elmore, Barnard, & Carr, 2007), as well as with poor mental and physical health in general (Heim, Ehlert, & Hellhammer, 2000; Jones et al., 2012; Steptoe et al., 2014), it is critical to understand predictors of cortisol AUCg in the postpartum period.

It is interesting to note that daily hassles were associated only with depression, and negative life events were associated only with cortisol AUCg. Although the current study had no a priori predictions of differences between the three chronic stress measures, previous research has shown that different types of stress differentially predict later depression (Pianta & Egeland, 1994). Economic stress did not predict any of the dependent variables. As previously noted, economic stress has been shown to interact with family support to predict cortisol AUCg in this sample (Jewell, Gress-Smith, Luecken, Crnic, & Gonzales, in press); it may be that the impact of economic stress on cortisol AUCg and other outcomes is largely dependent on moderating cultural or social factors such as family support. Additionally, although there was variability in economic stress as measured by the Economic Hardship Scale, it is important to note that all of the mothers in the study had family incomes below $25,000. It is likely that the majority of the women were undergoing significant economic hardship. Given the pervasive economic hardship in this sample, variations in economic stress among the mothers may have had small effects, which may have limited the current study's power to detect effects of economic stress on cortisol AUCg, depression, and self-rated health.

The current study has implications for mental health interventions in the postpartum period. Interventions aimed at reducing the impact of low-level, daily stressors may be successful in preventing later depressive symptoms. Additionally, prenatal daily hassle stress levels may be useful in identifying and targeting women most in need of support and intervention during this time period. As prenatal negative life events were shown to significantly predict postpartum cortisol levels, which in turn predict risk for later physical and mental health issues (Jolley et al., 2007; McEwen,

2000), interventions aimed at reducing negative life events (e.g., programs to reduce homelessness) or reducing the impact of negative life events on cortisol among low-income Mexican American women may be successful in promoting postpartum wellbeing.

The current study had several limitations. Due to the methodological constraints of the larger study, various measures were only available at certain time points. As levels of stress vary across pregnancy and into the postpartum period (Da Costa, Larouche, Dritsa, & Brender, 1999), measuring prenatal stress at only a single time point may not have revealed the full picture. Additionally, obtaining measures of pre-pregnancy cortisol would have allowed for controlling of baseline cortisol output, which would more strongly support the current interpretation that negative life events experienced during the prenatal period led to lower postpartum cortisol output. As the influence of perinatal hormonal changes on resting HRV is not well understood, the current study could have benefited from measuring resting HRV before pregnancy or using multiple measures of resting HRV to form a latent factor. Also, the current study only looked at chronic stress, heart rate variability, and three separate outcome measures (cortisol, depressive symptomatology, and self-rated health). Other measures that might be relevant to mental and physical health in the perinatal period, such as social support (Martinez-Schallmoser, Telleen, MacMullen, 2003; Sumner et al., 2011), acculturation (Martinez-Schallmoser et al., 2003), coping styles and behavior (Guardino & Dunkel-Schetter, 2014; Honey et al., 2003), and self-efficacy (Nierop, Wirtz, Bratsikas, Zimmerman, & Ehlert, 2008) were not the focus of the current study. Finally, the current sample included only low-income Mexican American mothers, the majority of whom primarily spoke

Spanish and were born outside of the U.S. The current results should not be generalized beyond this unique population.

Despite these limitations, the current study had many strengths. The current study utilized a prospective, longitudinal design that allowed for an investigation of the ways in which prenatal factors predict later postpartum outcomes. Additionally, the sample consisted of low-income Mexican American mothers, who are proportionally understudied in the health literature, despite being a quickly expanding population at high risk for poor postpartum outcomes. Furthermore, the investigation of resting HRV as a physiological moderator of prenatal chronic stress, as well as the use of objective and subjective measures of mental and physical postpartum health allows for an in-depth, biopsychosocial investigation of the ways in which prenatal chronic stress impacts postpartum health.

Future studies should continue to further investigate not only psychological and contextual buffers of the impact of stress on postpartum mental and physical health, but also other physiological indicators of resilience as possible protective factors during this time period (e.g. reactive HRV, sleep). Ideally, future studies will investigate multiple physiological, psychological (e.g. coping styles), and contextual (e.g. social support) buffers of chronic stress concurrently to gain a more comprehensive understanding of the processes involved in postpartum mental and physical health. Furthermore, future studies should try to understand the mechanisms through which daily hassles impact postpartum depressive symptoms- for example, by investigating the ways in which daily hassles may lead to dysfunctional cognitions or disruptions in sleep that then lead to postpartum

depressive symptoms. Similarly, the mechanisms through which negative life events leads to lower cortisol output are still not fully understood, and should be addressed through future research.

Summary and Conclusions

The current study assessed whether resting HRV moderated the impact of major and day-to-day chronic prenatal stressors on cortisol AUCg, depressive symptomatology, and self-rated health among Mexican American women in the postpartum period. Results did not support the hypothesis that high resting HRV would promote adaptive cortisol responses and levels, less depressive symptomatology, and better self-rated health in the face of chronic stress. However, a higher burden of prenatal daily hassles was associated with more postpartum depressive symptoms, and more prenatal negative life events predicted lower postpartum cortisol output. For Mexican American women, who are at higher risk for postpartum depression and poor postpartum health (Liu & Tronick, 2013), these stressors may significantly impair mental and physical health.

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Table 1.

Sample demographics

Prenatal marital status	N (%)	M (SD)	Range
Married or living w/partner	250 (77%)		
Not married or living w/partner	72 (23%)		
Education			
0 through 8 years of school	87 (27%)		
Some high school completed	104 (32%)		
High school graduate	86 (27%)		
Some college, vocational or technical school	26 (8%)		
College degree (BS/BA) or above	20 (6%)		
Number of children		2 (1.7)	1-10
Country of birth			
U.S.	44 (14%)		
Mexico	278 (86%)		
Family Income			
≤ $5,000	44 (14%)		
$5,001 - $10,000	61 (19%)		
$10,001 - $15,000	87 (28%)		
$15,001 - $20,000	37 (12%)		
$20,001 - $25,000	41 (13%)		
$25,001 - $30,000	16 (5%)		
$30,001 - $40,000	17 (5%)		
≥ $40,000	12 (4%)		
Language spoken at home			
English	58 (18%)		

Spanish	265 (82%)		

Table 2.

Descriptive Statistics of Study Variables

	N	Mean	SD	Range	Skew	Kurtosis
Prenatal Depressive Symptoms	322	6.13	5.53	0-25	.87	.16
Prenatal Self-Rated Health	321	3.27	1.05	1-5	.30	-.85
Daily Hassles	322	50.41	13.01	27-101	.70	.68
Negative Life Events	322	2.72	2.21	0-11	.90	.57
Economic Stress (z-score)	322	.01	3.0	-6.47-10.12	.35	-.14
11 wk mother AUCg[a] [b]	299	.80	.27	-.02-1.50	.07	-.25
11 wk Self-Rated Health[b]	310	3.60	.95	1-5	.05	-.69
Heart Rate Variability (RSA)	283	6.27	1.31	2.08-12.53	.64	2.20
12 wk mother AUCg[a]	192[c]	.77	.25	.08-1.33	-.17	-.21
12 wk Depressive Symptoms	308	3.86	4.36	0-18	1.17	.64
12 wk Self-Rated Health	203[c]	3.78	.99	1-5	-.32	-.57
18 wk mother AUCg[a] [b]	188[c]	.85	.25	.22-1.58	.23	.36
18 wk Self-Rated Health[b]	208[c]	3.69	1.02	1-5	-.27	-.60

[a] Log-transformed and weighted by ratio of individual's sampling time over mean sampling time

[b] Auxiliary variable, used for missing data analysis only

[c] Includes planned missingness

Table 3.

Zero Order Correlations

	1	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**	**11**
1. Daily Hassles	1.0										
2. Negative Life Events	.27*	1.0									
3. Economic Stress	.39*	.32*	1.0								
4. Heart Rate Variability	-.05	.02	.01	1.0							
5. Depressive symptoms (prenatal)	.56*	.35*	.34*	-.09	1.0						
6. Depressive symptoms (12 week)	.32*	.21*	.19*	-.05	.39*	1.0					
7. Self-rated health (prenatal)	-.30*	-.08	-.14*	.09	-.23*	-.20*	1.0				
8. Self-rated health (12 week)	-.15*	-.16*	-.12	-.08	-.10	-.23*	.29*	1.0			
9. Cortisol AUCg[a] (12 week)	.01	-.11	-.03	.07	-.06	-.02	.07	-.07	1.0		
10. Time of day[b]	-.01	-.01	.06	.04	-.1	-.03	-.06	.08	-.58*	1.0	
11. Mother's age	-.11	-.15*	.15*	-.17*	.01	.08	-.04	.06	-.05	.05	1.0

[a] Log-transformed and weighted by ratio of individual's sampling time over mean sampling time
[b] In minutes past midnight, for the 12 week home visit

Table 4.

Regression analyses: Cortisol AUCg

	B	*SE B*	*p-value*	Model R^2
(Constant)	1.507	.096	.001	.342
Daily Hassles	.001	.001	.760	
Heart Rate Variability	.017	.012	.145	
DH x HRV	.001	.001	.781	
Time of Data Collection	-.062	.006	.001	
Mother Age	.001	.002	.940	
(Constant)	1.521	.094	.001	.357
Negative Life Events	-.014	.007	.037	
Heart Rate Variability	.016	.012	.186	
NLE x HRV	.002	.005	.693	
Time of Data Collection	-.062	.006	.001	
Mother Age	.001	.002	.878	
(Constant)	1.481	.096	.001	.341
Economic Stress (z-score)	.001	.005	.770	
Heart Rate Variability	.016	.012	.164	
Econ x HRV	.005	.004	.227	
Time of Data Collection	-.060	.006	.001	
Mother Age	.001	.002	.816	

Note. All continuous variables centered prior to analysis.

Table 5.
Regression analyses: Depressive Symptomatology

	B	*SE B*	*p-value*	Model R^2
(Constant)	2.407	.381	.001	.168
Daily Hassles	.050	.020	.010	
Heart Rate Variability	-.040	.181	.810	
DH x HRV	.005	.011	.640	
Prenatal Depressive Symptomatology	.237	.05	.001	
(Constant)	2.140	.358	.001	.159
Negative Life Events	.153	.113	.174	
Heart Rate Variability	-.043	.182	.812	
NLE x HRV	-.087	.084	.305	
Prenatal Depressive Symptomatology	.281	.045	.001	
(Constant)	2.094	.356	.001	.155
Economic Stress (z-score)	.097	.082	.240	
Heart Rate Variability	-.045	.182	.806	
Econ x HRV	-.001	.064	.989	
Prenatal Depressive Symptomatology	.287	.044	.001	

Note. All continuous variables centered prior to analysis.

Table 6.
Regression analyses: Self-Rated Health

	B	*SE B*	*p-value*	Model R^2
(Constant)	2.909	.219	.001	.093
Daily Hassles	-.004	.005	.388	
Heart Rate Variability	-.064	.051	.208	
DH x HRV	-.004	.003	.248	
Prenatal Self-Rated Health	.260	.063	.001	
(Constant)	2.923	.212	.001	.094
Negative Life Events	-.047	.030	.108	
Heart Rate Variability	-.064	.051	.211	
NLE x HRV	.001	.024	.972	
Prenatal Self-Rated Health	.257	.061	.001	
(Constant)	2.923	.214	.001	.093
Economic Stress (z-score)	-.029	.021	.164	
Heart Rate Variability	-.063	.051	.215	
Econ x HRV	-.006	.017	.702	
Prenatal Self-Rated Health	.257	.061	.001	

Note. All continuous variables centered prior to analysis.